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THE BRITISH JOURNAL
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COMMUNICATIONS

THE OCULAR MANIFESTATIONS OF SPONTANEOUS SUBARACHNOID HAEMORRHAGE*

BY
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GLASGOW

SUBARACHNOID haemorrhage may occur spontaneously or as a result of head injury. The traumatic and non-traumatic cases present many points of similarity; but important differences result from the physical factors attending their onset.

The present communication is concerned with the non-traumatic group, and, especially, with the pathological findings in five cases which came to post-mortem examination. It is believed that these throw some light on the nature and mode of origin of the ocular complications.

Since the haemorrhage, in the majority of cases, comes from the vessels of the circle of Willis, being due to the rupture either of an aneurysm or of a weakened portion of the vessel wall, cases occur in which the effusion of blood is relatively slight, but prone to recurrence. The leading symptoms here, are severe deep-seated pain often accompanied by paresis of one or other of the ocular muscles. Cerebral arteriography may reveal the presence

* Received for publication, April 14, 1943.

of an aneurysm, and lumbar puncture discover blood staining of the cerebro-spinal fluid.

In the severe cases, such as those dealt with in this paper, the clinical picture is entirely different. The patient is seen in a more or less comatose state, with a history of sudden onset, and with signs suggestive of cerebral compression and meningeal irritation: pupil anomalies, nystagmus, conjugate deviation of the eyes, and paralysis of cranial nerves, including the oculomotors, are often observed. These are not in themselves sufficient to justify a diagnosis of subarachnoid haemorrhage. The presence of retinal haemorrhages and papilloedema, if these are found on ophthalmoscopic examination, strongly supports such a diagnosis, and a history of minor attacks adds to its probability, but the matter is settled by the discovery of blood in the cerebro-spinal fluid.

Such cases usually terminate fatally, and it is not surprising that in the majority no record is obtained of the state of the vision.

At post-mortem examination blood is found in large amount in the subarachnoid spaces at the base of the brain, sometimes breaking through into the substance of the brain or into the lateral ventricles. The source of the blood can usually be found in a ruptured aneurysm or a ruptured branch of the circle of Willis.

Immediately behind the optic canal, the optic nerves may show a bluish coloration owing to the presence of blood between the sheaths, and blood in this situation may be seen on the cut surface of the nerve. Even in cases where no blood is found in this region, exposure of the orbital part of the optic nerve often shows this to be expanded and discoloured by the presence of blood between its sheaths. In some cases (one of the five under discussion) this appearance affects the whole nerve from the optic foramen to the eyeball; but in the majority the ecchymosis is patchy and intermittent. It is usual to find some degree of bulbous distension of the optic nerve sheaths immediately behind the globe. Distension of the orbital veins, and haemorrhages amongst the orbital contents and on the surface of the sclera, may also be observed.

There has been an increasing interest in this subject in recent years, especially since the introduction of lumbar puncture.

Symonds (*1923*, *1924*), in reporting his personal cases, reviewed the bibliography, which then contained records of some 124 cases. His earliest reference is to *Wilks* (*1859*), but Wm. *Mackenzie* (1830) gives a clear description of the clinical and post-mortem appearance in a case described as one of "Amaurosis from dilatation of the cerebral arteries."

> "The sheath of the right optic nerve at its entrance to the eye was thickened and distended with blood, the veins on the

back of the sclerotic were much enlarged, and the ramifications of the retinal veins were readily seen all over the retina as far forward as the lens."

Throughout the literature attention has been concentrated on the question of the source of the blood which is found within the sheaths of the optic nerve and in the retina. It was natural to assume that the blood effused under pressure in the basal subarachnoid space passed through the optic canal by continuity into the subarachnoid space of the orbital part of the optic nerve, and thence into the retina or even into the vitreous (Tureen, 1941; Lyngar 1939).

That the blood in the sheaths of the optic nerve is of intracranial origin is still what may be called the "text-book" view; (Duke-Elder, 1940), but the investigations of Liebrecht (1906-1912) concerned chiefly with subarachnoid haemorrhage in injuries of the head, showed that the matter was by no means so simple. Several authors (Key and Retzius, 1870, Bartelli, 1938) cast doubts on the freedom of communication between the subarachnoid spaces of the brain and those of the optic nerve; but more recently Wolff (1940) has stated that, while in the upper part of the optic foramen the optic nerve, its sheaths and the periosteum are firmly bound together, there is, on the lower side, a clear channel between the intracranial and the orbital subarachnoid spaces.

Schieck (Discussion on Bücklers, 1935) held that if papilloedema and distension of the optic nerve sheaths, in cases of cerebral tumour, was due to the passage of cerebro-spinal fluid, the blood stained C.S.F. in subarachnoid haemorrhage could pass by the same route to the optic nerve sheaths and to the retina. This view, however, has no histological support. Liebrecht pointed out that sometimes blood was found in the sheaths of the optic nerve even when none was present in the subarachnoid space of the brain, that it was exceptional to find blood within the optic canal, and that at the apex of the orbit the perineural haemorrhage was not in the subarachnoid space of the nerve sheath, but in the dural sheath (for the most part surrounding the vessels in the sheath), passed forward by the lymph channels surrounding the vessels into the subdural space, and might remain entirely subdural or penetrate into the subarachnoid space.

He suggested three possible sources for the blood in the perineural subarachnoid space: (1) That in a few exceptional cases it might pass along the sheath through the optic canal. (2) That it might pass by the lymphatic spaces around the ophthalmic artery into the dural sheath and thence into the subarachnoid space. (3) That, in the majority of cases, blood from the torn vessels of the dural sheath passed into the dural tissue and thence

by pre-existent lymph channels into the subdural and subarachnoid spaces. As Liebrecht was dealing with traumatic cases, his third suggestion is not applicable to the cases of spontaneous haemorrhage.

Grimminger, at a later date (1924), made the suggestion that blood from the middle fossa may pass along the sheath of the first division of the 5th nerve, through the superior orbital fissure into the orbit, forming a haematoma of the ciliary nerves. It will be seen below that in one of my cases effusions of blood were found around the ciliary nerves and ciliary ganglion, but serial sections lend no support to the idea that this blood had tracked along the course of the 5th nerve.

Liebrecht, having found that, as a rule, there was no continuity between the blood in the middle fossa and that in the sheaths of the optic nerve, inclined to trace the latter to rupture of the vessels of the dural sheath within the optic canal. He did, however, point out (1906) that in histological examination of three cases the veins of the optic nerve, choroid, retina and other structures were found to be extremely full of blood, and that this was due to the sudden haemorrhage within the cranial cavity, and the consequent stasis of the blood in the ophthalmic vein and its branches. In several cases haemorrhage was found in direct relation to veins in the tissues peripheral to the lamina cribrosa, and Liebrecht expressed the view that the haemorrhages in optic nerve and retina occurred at the moment of the injury and were not secondary to that in the sheath.

To naked eye examination, only one of my own cases showed the optic nerve to be surrounded by blood in the canalicular segment. In two other cases serial sections in this region showed some blood corpuscles in the dural sheath; but this could not be taken as evidence of the passage of blood from the subarachnoid space of the brain to that of the optic nerve.

My own observations therefore support the view of Liebrecht, that while it may be a physical possibility for blood to pass through the optic canal in this way, it does so only in exceptional cases.

The occurrence of haemorrhage in the retina or in the vitreous has led to much speculation regarding the cause of this haemorrhage, and the source of the blood. There is no reason to believe that isolated haemorrhages in the retina could be derived from blood in the subarachnoid sheath of the optic nerve; but cases occur in which haemorrhage, sometimes massive, is found in front of the disc or crossing its borders. Such a haemorrhage is particularly liable to become subhyaloid, or to break through into the vitreous, and its clinical characters lend some colour to the view that the blood has tracked forward by the side of the optic nerve,

or along with the central vessels from the sheath spaces of the nerve. Uhthoff did not think that this was possible, and in this was supported by Dupuy Dutemps (1914), Riddoch and Goulden (1925) and others, but Leslie Paton (1924), as the result of personal observations, expressed his belief that such a leakage of blood could occur in a small percentage of cases. In this connection he reports two cases in which there was a large subhyaloid haemorrhage overlapping the optic disc and, in one case, subsequently breaking into the vitreous. Only one of these two cases came to necropsy. There was severe haemorrhage from a branch of the right middle cerebral artery extending into the right lateral ventricle, the basal subarachnoid space and the sheaths of both optic nerves. Paton's paper does not state that any histological examination was made to establish the connection, if any, between the perineural haemorrhage and that in the retina, and so far as I am aware, there has been no microscopic proof of the passage of blood from the optic nerve sheaths to the retina in the way assumed by Paton for his cases. The more generally accepted view is that distension of the subarachnoid space of the nerve by the effused blood causes obstruction of the blood flow in the central retinal vein where it crosses the subarachnoid space, in a very smilar way to the condition seen in thrombosis of the central vein. Paton sees the difficulty of reconciling this explanation with the very different appearance of the retinal haemorrhages in the two conditions. But the circumstances under which the retinal haemorrhage occurs in thrombosis of the central vein and in subarachnoid haemorrhage are very different. In the former, although the final block may be somewhat sudden, there has been a long period of progressive change in the affected vein. There has been increasing narrowing of its lumen with a gradually increasing impediment to the venous circulation and consequent changes in the permeability of the walls of the retinal branches. In the case of subarachnoid haemorrhage, on the other hand, whether traumatic or spontaneous, there may be no antecedent cause for such a general weakening of the walls of the retinal veins, and the stasis is sudden and severe.

As I have already mentioned, Liebrecht found, in histological examination of three cases, that the veins of the choroid, retina, optic nerve and other structures were extremely full of blood; and attributed this to the sudden rise of intracranial pressure with consequent stasis of the blood in the ophthalmic vein and its branches.

The sudden rise of intracranial pressure here referred to, and the influence of the consequent venous stasis on the occurrence and distribution of the haemorrhages throughout the orbital and ocular structures was also alluded to by Bücklers (1935), but

seems to have received no further comment, although it would seem to be the most important single factor in producing the clinical picture of subarachnoid haemorrhage from the ophthalmic point of view. It is not necessary to deny that stasis and haemorrhage may be caused by sudden pressure or tension on the central vein where it crosses from the optic nerve to its sheaths; but if this does occur it can only explain the haemorrhages peripheral to that point.

It should be noted, moreover, that distension of the sheaths by blood is not invariably present; indeed it only occurred in one of my five cases; in the other four there was not even evidence that the central vein had been compressed by haemorrhage. In these four cases the distension of the sheaths was caused by fluid in the subarachnoid space, and the fact that the ampulliform swelling of the nerve ceased some 3 or 4 mm. in front of the crossing of the vessel does not suggest the occurrence of tension on the central vein. Again, the assumption of pressure or tension on the central vein as the cause of the retinal haemorrhage takes no account of the fact, which emerges from the cases here reported, that haemorrhages occur simultaneously at many points outside of the field of distribution of the central retinal vein; and one can only reach a satisfactory explanation of all the ocular phenomena in this condition by assuming a sudden venous obstruction affecting a much wider area.

Illustrative Cases

This communication deals with five fatal cases of subarachnoid haemorrhage which were submitted to post-mortem examination, and in which sections were obtained of the eyes and optic nerves. The cases differ to some extent in detail, but illustrate all the characteristic features of this condition. Four of the five were women, and one a man, the ages ranging from 39 to 62. All had elevation of blood pressure, severe in three and moderate in two. In only one were the fundi free from haemorrhage. In four the source of the subarachnoid haemorrhage was discovered; in one it came from a rupture of the middle cerebral artery, and in three from rupture of an aneurysm of the vessels of the Circle of Willis.

CASE 1.—A woman aged 50 years. Malignant hypertension, B.P. 230/150. W.R. positive. Gave a history of having had double vision, but no motor defect on examination. C.S.F. blood-stained. O.E.R. General pallor and opacity of fundus due to diffuse exudate. Clusters of white specks at the macula. Arteries very narrow, with marked variation of calibre. Thrombosis of a secondary venous branch with formation of network of collaterals. Flame-shaped haemorrhages. White woolly patches.

O.E.L. Condition somewhat similar, but no venous thrombosis.

Death. Post-mortem examination: Extensive subarachnoid and intracerebral haemorrhage probably from right middle cerebral artery. Bulbous expansion of both optic nerves behind the globe. Bluish discolouration due to haemorrhage within the sheath.

Sections of the retina showed the characteristic changes of hypertensive retinopathy: ganglioform degeneration in the nerve fibre layer, masses of eosin-stained exudate in the internuclear layer and behind the layer of rods and cones, haemorrhages in all the retinal layers, and between the neuro-epithelium and pigmented layer.

The larger arteries in the retina showed little or no change. The walls of the capillaries and smaller arterioles were thickened and the lumen round and open. Thickening of the vein walls was also seen.

Serial sections of the right optic nerve (and sections of the left optic nerve presented no essential difference), from behind the optic foramen to the anterior extremity of the nerve, demonstrated the distribution of haemorrhage within the sheaths. Blood was found throughout the whole length of the nerve, but varied in situation and in quantity from point to point. Thus: in the canalicular segment there were two minor effusions of blood (one of them very small), within the dural sheath, and a few red blood corpuscles in the pia-arachnoid (Fig. 1). Further forward in the same segment the blood in the dura was very scanty and that in the pia-arachnoid relatively increased. In this part of the nerve there was no subdural haemorrhage linking that in the dura with that in the pia-arachnoid. Passing forward the haemorrhage increased, till, coming to the apex of the orbit, we found several massive haemorrhages in the dural sheath, and relatively slight haemorrhage in and under the arachnoid (Fig. 2). At some points these dural haemorrhages seemed to be closely related to the vessels.

Behind the point where the central vessels cross from the dural sheath to the nerve, there was an increased amount of subarachnoid haemorrhage and only a few corpuscles outside of the arachnoid (Fig. 3). Throughout the next segment of the nerve, which included the central retinal vessels in their passage across the vaginal spaces, there was no massive haemorrhage, but scattered clusters of red cells both subdural and subarachnoid in position. Where the central vein lies in the dura, just before crossing the space (Fig. 4) there was some blood lying close to it at one side, but nothing to suggest compression of the vein; and the artery and vein were not accompanied by blood at any other part of their course. Further forward the blood became more scanty; still found

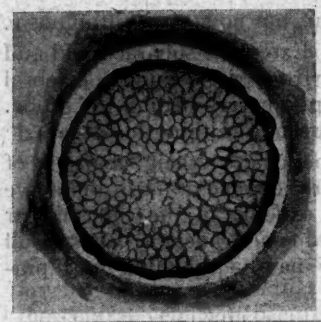


Fig. 1

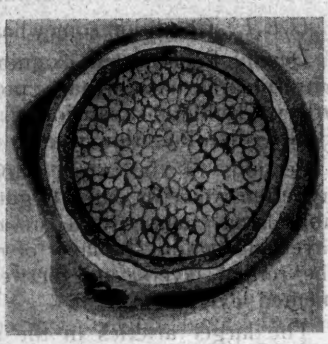
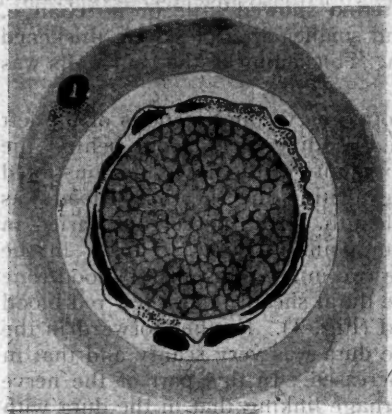


Fig. 2



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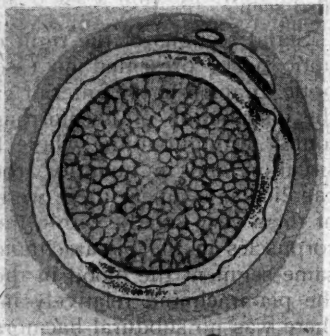

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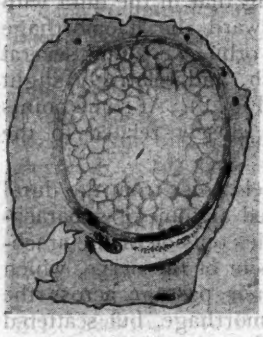

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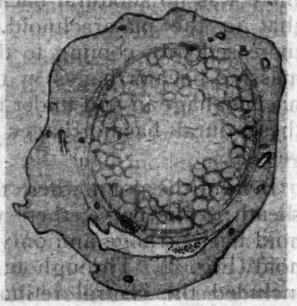


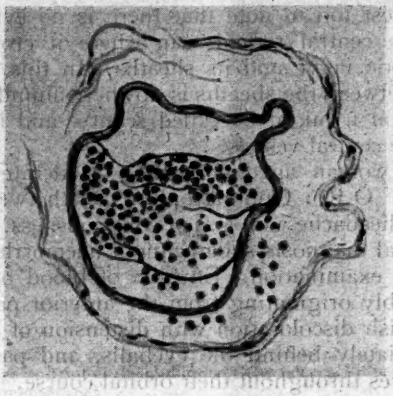
Fig. 6

CASE 1.—FIGS. 1 to 6.

Transverse sections of R. optic nerve. 1. The canalicular segment. Light haemorrhage in dural sheath and pia-arachnoid. 2. At apex of the orbit. Massive haemorrhages in dura, light in pia-arachnoid. 3. Behind entrance of the central retinal vessels. Haemorrhage mainly subarachnoid. 4. Where central vessels lie in dural sheath. Light haemorrhage, dural, subdural and subarachnoid. 5. Near anterior end of vaginal space (section somewhat oblique). Scanty haemorrhage in dural and pial sheaths, and in space. 6. At level of lamina cribrosa. Scanty haemorrhage in space and in border tissue.

in the subdural and subarachnoid spaces. Where the vessels reached the centre of the nerve there was again an increase in the quantity of blood, this time entirely subdural. Near the anterior end of the intervaginal space there was again a slight increase in the haemorrhage in the dura, subdural and subarachnoid, and in the pial sheath (Fig. 5). At the level of the lamina cribrosa there were merely a few clusters of erythrocytes in the dense tissue which separates the nerve from the anterior extremity of the intervaginal space (Fig. 6).

Antero-posterior sections through the optic nerve entrance and the neighbouring retina showed haemorrhage under the neuro-epithelium apparently tracking forward, via the " border tissue "



CASE 1.—FIG. 7.

Vein on outside of dural sheath. Dissecting haemorrhage between endothelium and media and between media and adventitia.

into the retina, in a situation corresponding with the marginal haemorrhage seen ophthalmoscopically.

The segment of the nerve behind the optic canal included the internal carotid artery and the ophthalmic artery. The carotid at this point was grossly atheromatous, with calcification and fatty changes in the atheromatous patches, and breaking down and calcification of the internal elastic lamina. The ophthalmic artery at its point of origin from the carotid also showed some thickening of the intima and calcification of the elastic lamina; but throughout the rest of the sections the ophthalmic artery, the central vessels and the vessels of the sheaths were practically normal.

Comment

The changes in the retina have probably no bearing on the subject of subarachnoid haemorrhage, being those found characteristically in hypertensive retinopathy: but the sheath haemorrhages are definitely part of the picture of subarachnoid haemorrhage. The points of special interest are: (1) the wide distribution of the haemorrhage throughout the whole course of the optic nerve, intracranial, canalicular and orbital, and (2) the fact that the haemorrhage is patchy in its occurrence, and varies in its intensity and in its situation. There is no continuity between the effusions of blood in the dural sheath and those in the subdural and subarachnoid spaces, and we are obviously dealing with a number of discrete haemorrhages, not with blood tracking along the sheaths from the middle fossa to the anterior extremity of the nerve.

It is of interest too to note that there is no evidence of compression of the central retinal vein where it crosses the space between the optic nerve and its sheaths. In this particular case haemorrhage between the sheaths is nowhere abundant, and at the point in question it might be called scanty, and it is practically unrelated to the central vessels.

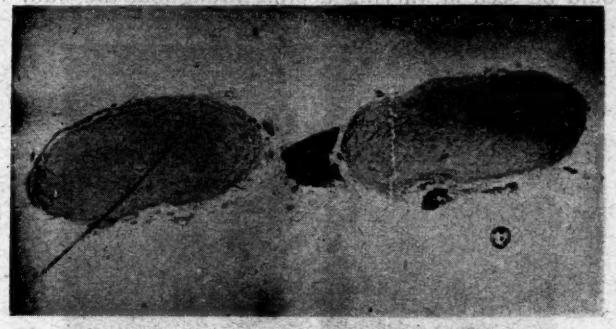
CASE 2.—A woman aged 56 years. Moderate hypertension. W.R. positive. O.E.: Optic atrophy. Death twelve hours after onset of severe headache with loss of consciousness. C.S.F. blood-stained. Clinical diagnosis subarachnoid haemorrhage, confirmed at post-mortem examination. Effusion of blood basal and intracerebral, probably originating from the anterior part of the circle of Willis. Bluish discoloration with distension of the optic nerve sheaths immediately behind the eyeballs, and patchy discoloration of the nerves throughout their orbital course. A small ecchymotic patch in both nerves just behind the optic canal. A haemorrhage of pin-head size on the anterior aspect of the chiasma.

Anteroposterior sections of the retina, choroid, sclera and optic nerve of the right eye showed no haemorrhages in these structures, but small discrete haemorrhages in and beneath the dural sheath, under the arachnoid and in the pial sheath. The subarachnoid haemorrhage was scanty, and the notable distension of the sheath must have been caused by fluid. It was observed also that some of the vessels of the retina were surrounded by a clear perivascular space, more pronounced in the veins than in the arteries. Since the veins as a whole were well filled with blood and the arteries empty, the perivascular space could not be looked upon as an artefact. It apparently indicated a stasis of fluid analogous to that within the nerve sheaths. The optic nerve itself was atrophic, with thickening of the connective tissue septa.

Similar sections in the left eye also showed a pronounced distension of the subarachnoid space apparently due to fluid.

Only a few small clusters of red blood corpuscles were found in the anterior part of the space. Anterior to the point of emergence of the central vessels there was some subdural haemorrhage, with scanty subarachnoid haemorrhage here and in the anterior extremity of this space. In the neighbourhood of the vessels themselves, crossing the space, haemorrhage was very scanty, and still more so behind this point. No haemorrhages were found in the retina or choroid or in the optic nerve itself.

The central retinal artery and vein or their branches in the retina, presented little abnormality other than the enlargement of the perivascular lymph space referred to, but among the vessels



CASE 2.—FIG. 8.

Transverse section of optic nerves in front of chiasma. Large haemorrhage in front of the junction of the nerves, diffuse haemorrhage in the soft membranes.

on the outer aspect of the dural sheath there were arteries with thickened hyaline-looking walls and veins with lymphocytic cuffing.

The presence of a small haemorrhage on the anterior aspect of the chiasma had attracted notice at the *post-mortem* examination (Fig. 8), and the chiasma with stumps of the optic nerves and optic tracts was fixed and cut serially in paraffin. In addition to the compact haemorrhage between the optic nerves in front of the chiasma, the sections showed a good deal of diffuse haemorrhage in the meningeal covering of the chiasma and nerves. The walls of the vessels of the arachnoid were feebly stained with haemalum, possibly owing to a *post-mortem* change; but, buried in the principal haemorrhage, in this situation, there was an incomplete ring of endothelial cells representing the wall of such a vessel, and probably the site of the rupture which was the source of this haemorrhage. Among the vessels of the pial and arachnoid

sheaths several were found to have red blood corpuscles between the layers of their walls, and in the perivascular space.

Within the chiasma and the attached portions of the optic nerves, the smallest vessels presented thick hyaline-looking walls and a small lumen (about 14 to 40μ) and appeared as rigid as pipe stems. At more than one point vessels of the pia and of the arachnoid showed a breach in their wall through which blood had apparently escaped; but more interesting was the discovery, in the substance of the chiasma and optic tracts, of many vessels showing haemorrhage within and outside of their walls (Figs. 9-14). In some cases this amounted merely to one or two red blood corpuscles between the layers of the wall, in others many corpuscles could be seen at varying depths throughout the wall, while

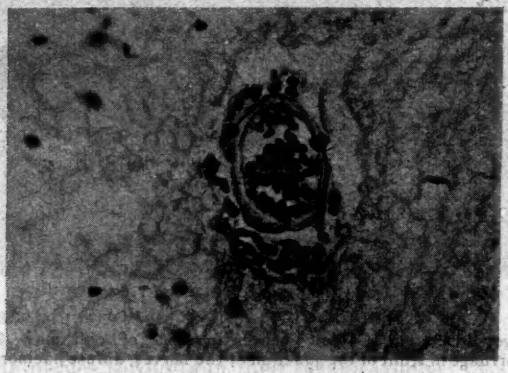


FIG. 9.

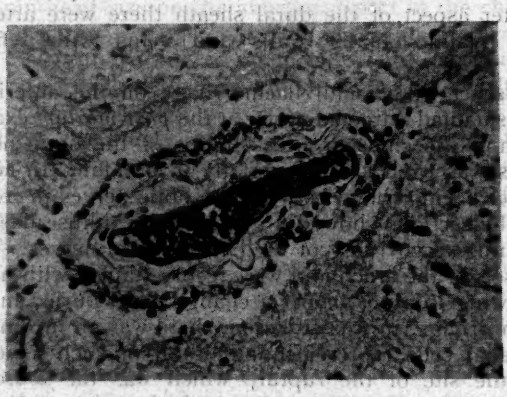


FIG. 10.

FIG. 11.

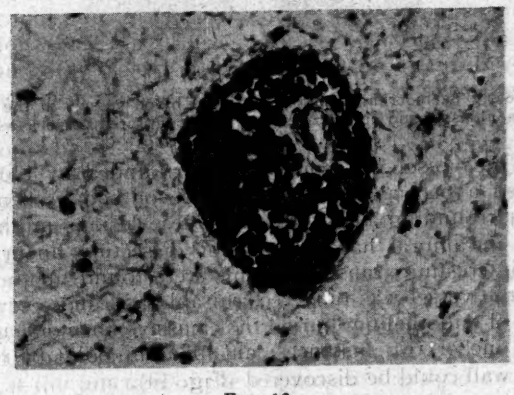


FIG. 12.

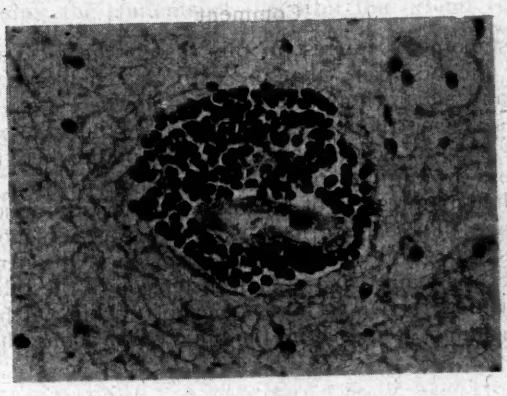
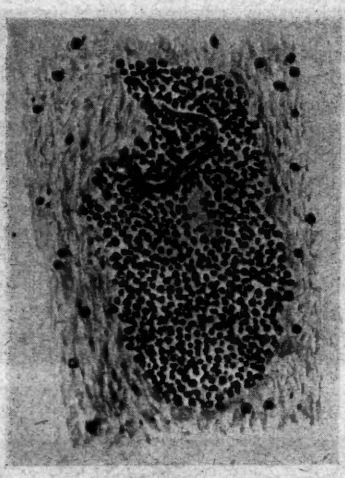


FIG. 13.



CASE 2.—FIGS. 9 to 14.

Dissecting haemorrhages in the walls of blood vessels in the optic nerves, chiasma and optic tracts. In 14 the ruptured vessel is visible.

in others the vessel was completely surrounded by blood which either filled a perivascular lymph space or had broken through into the surrounding nerve tissue. These lesions might be described as intramural haematomata or dissecting aneurysms. A series of interesting transition forms were found up to the stage at which the vessel was only recognisable as a more or less complete tube of endothelium buried in a massive haemorrhage. Here again, in one or two instances, an apparent point of rupture of the vessel wall could be discovered (Fig. 14).

Comment

The first point of interest in this case is the fact, already recorded in the first case, that the extravasated blood within the subdural and subarachnoid spaces, was nowhere very abundant. The largest haemorrhage was found in the subdural space at one side of the series of sections anterior to the point where the central vessels cross the vaginal spaces, and there was nothing to support the idea that compression of the vessels could be produced by the haemorrhage. The haemorrhage, such as it was, was mostly in the subdural space: while the notable expansion of the nerve behind the eyeball was mainly due to fluid distension of the subarachnoid space. Further, there was no evidence of tracking of blood from the subarachnoid space behind the optic canal, along the nerve to

its anterior extremity. The haemorrhages in different situations were discrete, and there was at least some evidence of rupture of vessels at a number of points within the dura and elsewhere. Finally, the unexpected finding of isolated haemorrhages from vessels of the membranes covering the chiasma, and still more unequivocal haemorrhages from vessels in the chiasma and optic tracts, shows that the familiar hypothesis of compression of the central vessels, offered to explain the occurrence of the retinal haemorrhages, is quite inadequate when we come to consider the wider pathological picture.

This was the only one of our cases in which investigation extended beyond the optic nerve, the eyeball and the orbital structures. The significance of the haemorrhages in the chiasma and optic tracts is therefore uncertain, but if, on further enquiry, it can be shown that such haemorrhages occur, for example, within the brain stem, they would provide a reasonable explanation of the oculomotor and other palsies which are such a frequent feature of the condition (Case 4). Such a sign as conjugate deviation of the head and eyes (see Case 5) cannot be satisfactorily explained by direct action of the intracranial haemorrhage on nerve trunks.

CASE 3.—A woman, aged 62 years. Unconsciousness of sudden onset 2½ hours before admission to hospital. Twitching of hands, arms and face. Vomiting. B.P. 226/138. Blood in C.S.F. *O.E.* R.E. large recent haemorrhage in lower temporal quadrant of fundus, and other smaller ones elsewhere. L.E. A few small haemorrhages.

Death occurred within 24 hours. *Post-mortem* examination showed gross subarachnoid haemorrhage over the base of the brain, as well as haemorrhage in the lateral ventricles, and in the left frontal lobe. The source of the blood was the rupture of an aneurysm at the junction of the left anterior cerebral and anterior communicating arteries.

On exposing the contents of the orbit the orbital veins were found to be markedly distended. There were haemorrhages among the orbital fat, in Tenon's capsule, on the posterior surface of the eyeball, on the outer surface of the dural sheath, and within the sheaths of both optic nerves from the optic canal to the sclera. Naked eye examination of the retina confirmed the ophthalmoscopic appearances. Papilloedema was discovered both on slit-lamp examination and in sections.

Microscopic examination showed the severe and widespread character of the bleeding in this case. On the right side there was abundant effusion of blood filling the vaginal spaces along the course of the optic nerve, from its point of contact with the internal carotid artery to the back of the eyeball, at all depths: dural, subdural and subarachnoid; and in the anterior part so

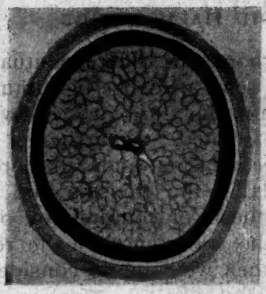


Fig. 15

Fig. 16

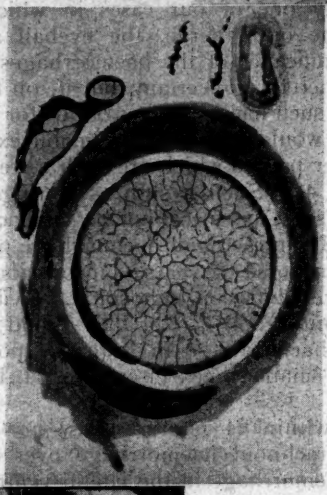


Fig. 17

Fig. 18

Fig. 19

CASE 3.—FIGS. 15 to 19.

Transverse section of right optic nerve. 15. Near anterior end of nerve. Blood almost completely filling the space. 16. Near apex of orbit. Massive haemorrhage in dural sheath breaking through into subdural and subarachnoid spaces. 17. Slightly anterior to 16. Haemorrhage among the orbital tissues. 18. Haemorrhage surrounding ophthalmic artery, in the dural sheath, and in the sheath spaces, embracing the central retinal vessels. 19. Behind the entrance of the central vessels. Haemorrhage in the pia-arachnoid, and overfilling of vessels of the pial septa.

massive that the arachnoid membrane could not be traced. There was also massive haemorrhage among the fibres of the dural sheath, and on its outer surface. In the posterior third of the nerve there was massive haemorrhage, breaking through into the subdural and subarachnoid spaces. But, in addition to these effusions in the usual situations, blood was found within the optic canal, outside the dural sheath at the apex of the orbit (Fig. 17), and, further forward, in the pial sheath (Fig. 18). There was

CASE 3.—FIG. 20.

Antero-posterior section of right optic nerve. Massive haemorrhage fills the spaces and distends the sheaths. There are haemorrhages at all depths in the retina, before and behind the retina, in the sclera and round the vessels of the Circle of Zinn.

haemorrhage surrounding part of the ophthalmic artery, in the dural sheath where penetrated by the central artery and vein, and in the pia surrounding the central vessels (Fig. 18).

About 15 mm. behind the entrance of the central vessels there was at one side of the nerve a notable distension of the vessels of the pia, and of the septa of the nerve, and diffuse haemorrhage in the adjacent part of the pia and arachnoid (Fig. 19). In the anteroposterior sections at the optic nerve entrance (Fig. 20) haemorrhage was found surrounding the vessels of the circle of Zinn, in the sclera, among the nerve fibre bundles in front of the lamina cribrosa, in all the layers of the retina, in front of the disc, in the vitreous, and in front of, and beneath the retina.

In the intracranial part of the nerve there was patchy haemorrhage dural, subdural, subarachnoid, and in the pial sheath.

CASE 3.—FIG. 21.

Antero-posterior section of left optic nerve. Massive haemorrhage fills the spaces, and there is diffuse haemorrhage in the epibulbar tissues near the optic nerve entrance.

The internal carotid artery showed atheroma with calcification, and in the adjacent portion of the optic nerve there was hyperaemia in the septal vessels and a good deal of haemorrhage in the substance of the nerve itself.

In the left eye no retinal haemorrhages were seen in the sections passing through the papilla; but the sheath haemorrhage was almost as abundant as in the right eye. At the anterior end of the nerve the sheath spaces were completely filled with blood as far as the cul-de-sac, and haemorrhages were found among the fatty and connective tissues in the angle between the dural sheath and the sclera. It was interesting to observe that the central retinal vein, in its course through the nerve, was not well filled with blood, and in this situation there was no undue filling of the vessels of the septa of the nerve.

In both eyes there was unequal filling of the choroidal vessels; in some parts this seemed to be excessive. Throughout both the retinal and the ciliary vessel systems the arteries showed medial sclerosis and some thickening of the intima, and some of the veins had thickened walls. The arteries of the choroid were markedly sclerosed, and the veins here also had thickened walls.

Comment

This case, like the two preceding cases, shows in the first place that we are not dealing with the simple question of intracranial subarachnoid haemorrhage passing along the subarachnoid

space of the optic nerve sheaths, through the optic canal and along the orbital portion of the nerve. We found a striking venous congestion in the orbital venous system and haemorrhages in the orbit, in all the sheaths of the nerves, and in the optic nerves themselves. In the more markedly affected right eye there was haemorrhage in all the layers of the retina, as well as behind and in front of the retina, and in the vitreous.

It is noteworthy that of the five cases here reported, this was the only one in which we could say that the sheaths of the optic nerves were *distended* with blood, and even here we had no evidence that compression of the central retinal vein was the cause of the retinal haemorrhage.

CASE 4.—A woman aged 39 years. Essential hypertension of at least 8 years duration. No nephritis. Father died 2 weeks before her admission. Injury to face and side five days before admission. Headache, vomiting, confusion and drowsiness for 24 hours before admission. B.P. 238/116. Albuminuria. R. hemiparesis. Paresis of L. 3rd and 4th cranial nerves. Blood in C.S.F. W.R. negative. *O.E.* Many white woolly patches and haemorrhages. The latter of varying size but mostly large, dark and of rounded form. *Post-mortem* examination : Subdural haemorrhage in small amount in both anterior fossae. Marked subarachnoid haemorrhage in neighbourhood of circle of Willis, some of it of apparently old standing. Ruptured aneurysm at junction of basilar and posterior cerebral arteries. Haemorrhage into the superficial layers of the pons, especially to the left of the middle line.

Marked distension, and blue discoloration of both optic nerves behind the globe. Ecchymosis of the nerve sheaths at one or two discrete points between the globe and the optic canal, but no naked eye evidence of blood within the sheaths behind the optic canal.

Sections of the right retina showed gross haemorrhages in the nerve fibre, and outer molecular layers. For the most part these seemed to be quite independent of each other, the inner nuclear and inner molecular layers being unaffected; but at several points the haemorrhage had broken into the intervening layers. In the same region the outer molecular layer, over a wide area, was occupied by an amorphous (albuminous?) coagulum.

Serial sections of the right optic nerve showed, at the level of the lamina cribrosa, some scanty haemorrhage in the border tissue and in the inner layers of the dura at the anterior extremity of the subdural space. Absence of other haemorrhage was conspicuous in the first two or three millimetres of the nerve.

Between 3 and 6 mm. behind the globe the maximum distension of the sheath was situated. This was for the most part due to effusion of fluid in the subarachnoid space, which contained very

few blood corpuscles, while the subdural space over the same segment of the nerve contained several gross patches of haemorrhage which were evident to the naked eye (Fig. 22). Passing backwards to the point of entrance of the central retinal artery, the sheath haemorrhage became progressively less, until, at the level where the vessels first appeared at the centre of the nerve, there was a mere trace of blood in the subdural space (Fig. 23). No significant haemorrhage was found in association with the central vessels at the point of crossing or elsewhere.

The central retinal vessels and those of the pial sheath showed slight medial sclerosis.

Sections of the optic nerve and surrounding tissues in the posterior part of the left orbit, showed only traces of haemorrhage in the subdural space and still less in the subarachnoid, but there were a number of haemorrhages in the dural sheath, and several of these surrounded or were closely associated with thin-walled vessels of the sheath. Some diffuse haemorrhage was found among the bundles of the orbital muscles, and in the connective tissue framework of the orbital fat. Still more interesting was the discovery, in a region where there was practically no blood in the

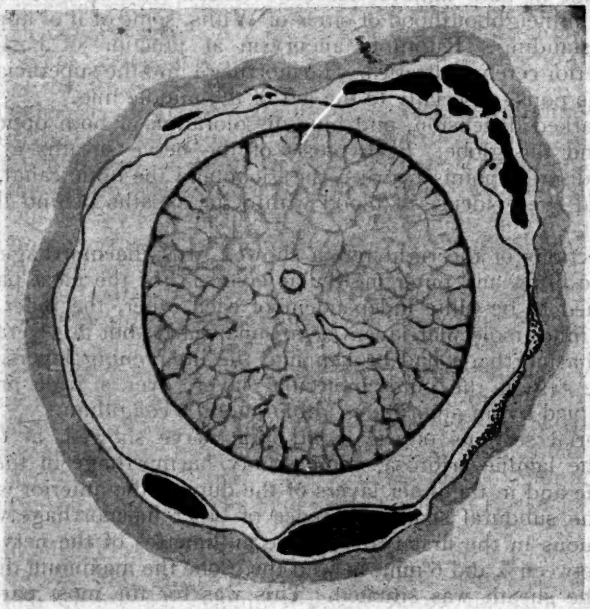


FIG. 22.

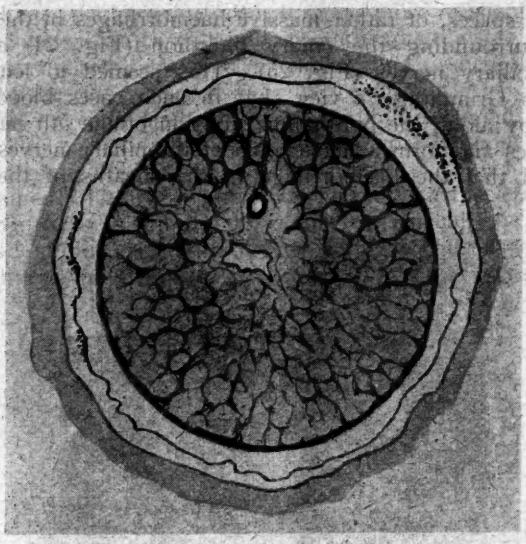


FIG. 23.

CASE 4.—FIGS. 22 and 23.

Transverse sections of the right optic nerve. 22. A few mm. behind the globe. Massive haemorrhages in subdural space, and distension of subarachnoid space by clear fluid. 23. At the level of the entrance of the central vessels. A mere trace of haemorrhage in the subdural space.

CASE 4.—FIG. 24.

Haemorrhage in connective tissue sheath of ciliary ganglion.

sheaths or spaces, of rather massive haemorrhages in the fibrous sheaths surrounding the ciliary ganglion (Fig. 24) and the posterior ciliary nerves (Fig. 25). This seemed to lend some support to Grimminger's view that in such cases blood might find its way along the ophthalmic division of the 5th nerve and its branches and form a haematoma of the ciliary nerves; but it was found that the haemorrhage in the sheaths of the ciliary

CASE 4.—FIG. 25.

Haemorrhage in connective tissue sheath of posterior ciliary nerves.

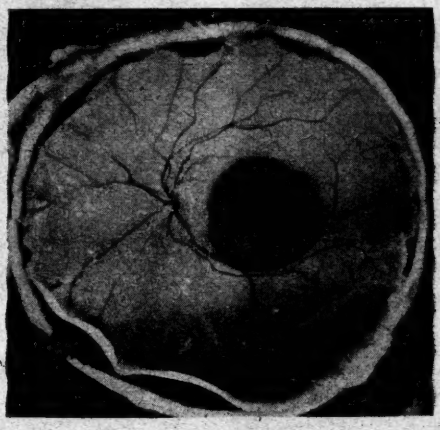
nerves was at its minimum in the posterior part of the orbit and increased in amount further forward. The reverse would be expected if the blood were tracking forward. Moreover, the blood surrounding both the ciliary nerves and the ciliary ganglion was confined to the fibrous sheaths of these structures, and was absent from the space beneath the sheaths.

Comment

In this case, as in all but one of the group, there was no massive haemorrhage between the sheaths of the nerve, and such as it was the haemorrhage was mainly in and beneath the dural sheath. There was nothing to suggest the view that the central retinal veins were compressed by haemorrhage within the spaces. The subarachnoid space, on the other hand, was, anteriorly, greatly distended by fluid. If this were sudden in its onset it could conceivably cause both pressure and tension on the vein, as it traversed the sheaths after leaving the nerve, that might cause the sheath haemorrhage anterior to this point as well as the

haemorrhages in the retina. But it seems more reasonable to seek for a mechanism which will embrace and explain at the same time the haemorrhages discovered in the orbital tissues and in the sheaths of the ciliary nerves and ciliary ganglion.

CASE 5.—A man aged 50 years. Admitted to hospital in a state of coma, having collapsed at his work without warning. Blood pressure was 188/98. W.R. negative. The C.S.F. was blood-stained. The right pupil was larger than the left, and unresponsive to light stimuli, direct and consensual, the left pupil contracted slightly to light directly but not consensually, there was slight drooping of the right upper lid and there was paralysis

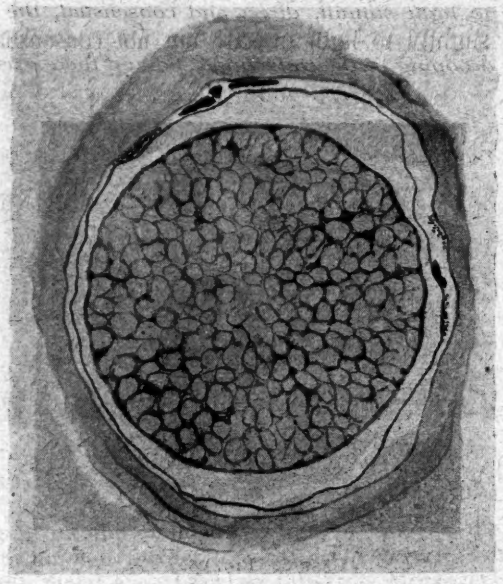


CASE 5.—FIG. 26.

Subhyaloid haemorrhage in the left fundus. Post-mortem photograph.

of conjugate movement of the head and eyes to the left. O.E. Moderate papilloedema and fulness of the retinal veins in both eyes, and in the left a large subhyaloid haemorrhage occupying the centre of the fundus (Fig. 26). No other haemorrhages discovered. Death 2 weeks after admission. *Post-mortem* examination confirmed the diagnosis of subarachnoid haemorrhage, the source of the blood being a ruptured aneurysm at the junction of the anterior cerebral and anterior communicating arteries. The optic nerves presented a bluish-coloured bulbous expansion behind each globe, and irregular discoloration as far back as the optic canal. Naked eye and slit-lamp examination of the fundi revealed no lesions other than the subhyaloid haemorrhage, and in sections of the left eye only one other small retinal haemorrhage was found. The retinal vessels, especially on and near the disc,

were well filled and many capillaries could be traced by their blood content. No pathological changes were found in the retinal vessels. In many of the transverse sections of the left optic nerve, especially behind the point of entry of the central vessels, there was only scanty haemorrhage; this was entirely subdural in position and showed discontinuity both in the length and in the circumference of the nerve (Fig. 27).



CASE 5.—FIG. 27.

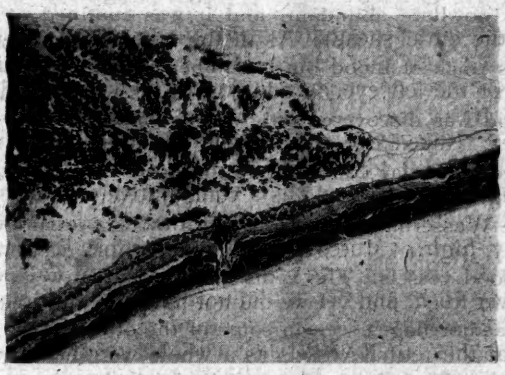
Transverse section of left optic nerve. Scanty subdural haemorrhage and fluid distension of sub-arachnoid space.

In front of this point there was an increase in the amount of haemorrhage, perhaps mainly in the neighbourhood of the emerging vessels; but the blood had no close relation to these vessels, nor could one trace any haemorrhage accompanying the vessels in their course into or through the dural sheath.

At this level, but a little distance from the central vessels, there was a small independent haemorrhage between the lamellae of the dural sheath. Some distance behind this point, where the central artery and vein were emerging from the dural sheath to join the ophthalmic artery and vein, there was haemorrhage in the adventitia of both vessels, especially of the vein. At the

anterior extremity of the optic nerve there was a compact layer of haemorrhage on the inner surface of the dural sheath, but none in the widely dilated subarachnoid space.

The subhyaloid haemorrhage was definitely in front of the internal limiting membrane of the retina, its anterior aspect covered by a thin membrane which at some points showed continuity with delicate membranes or filaments in the vitreous (Fig. 28). The apparent source of this haemorrhage was a well filled vein coming up to the retinal surface, whose blood content seemed to be in continuity with the subhyaloid extravasation (Fig. 29).



CASE 5.—FIG. 28.

Retina and subhyaloid haemorrhage, left eye.

CASE 5.—FIG. 29.

Showing probable source of subhyaloid haemorrhage.

The picture of the fundus of this eye shows that the lower temporal vein, lies deep to the subhyaloid haemorrhage near its lower border, and the microscopic sections show that the vessel above referred to as the possible source of the haemorrhage is a venous branch which joins the inferior temporal vein under cover of the subhyaloid haemorrhage about 3 mm. temporal to the fovea.

Comment

A notable point in this case is the limited extent and distribution of the bleeding. Haemorrhage along the course of the nerve was nowhere abundant, and it was confined to the inner aspect of the dural sheath. As in the other cases, there was no continuous track of blood but a number of discrete effusions. In the retina of the left eye, in addition to the subhyaloid haemorrhage, blood was discovered only at one small point in the nerve fibre layer. Naked eye and slit-lamp examination of the right eye showed a similar condition of the optic nerve and no retinal haemorrhage. The patient was a comparatively young man with a negative Wassermann reaction, and a blood pressure which was not unduly high. Moreover, the microscopic sections showed healthy blood vessels. The veins and capillaries were well filled, perhaps over filled, and yet we did not have the multiple and more massive haemorrhages seen in some of the other cases. We must believe that the retinal vessels as a whole were healthy, but that there were at least two points of diminished resistance in their walls. One wonders whether these were the ocular counterparts of the weakened walls which allow the formation and rupture of aneurysms on the cerebral vessels.

In this case there was no blood in the subarachnoid space of the optic nerve sheath and therefore there is no question of tracking of blood from the cerebral subarachnoid space. We can assert, too, that neither the subdural haemorrhages, nor those in the dura and accompanying the central retinal vessels where they are leaving the dural sheath, can be explained by pressure of blood on the central vein in the perineural space. It is equally certain that the preretinal haemorrhage did not arise by blood passing along the course of the central vessels or by the side of the optic nerve into the retina.

General Comment

The five cases here reported present a certain similarity in their general character; the history of sudden onset with severe pain, the symptoms of cerebral irritation and compression, and coma preceding death. Blood-staining of the cerebro-spinal fluid was, of course, present in all.

They differ more widely in the eye conditions clinically observed: the presence or absence of oculomotor paralysis, and disturbances of conjugate movements, of papilloedema, and of haemorrhage on the optic disc, at different depths in the retina, in front of the retina (subhyaloid) and in the vitreous. There were also certain fundus changes which did not result from the intracranial haemorrhage but were referable rather to a constitutional state which might be the cause of the haemorrhage, as, for example, the picture of hypertensive retinopathy in the first case, and of optic atrophy, probably syphilitic, in the second.

Coming to the pathology of the cases we find a striking and quite unexpected variety in the distribution of the haemorrhage and, incidentally in the state of the ocular and other blood vessels.

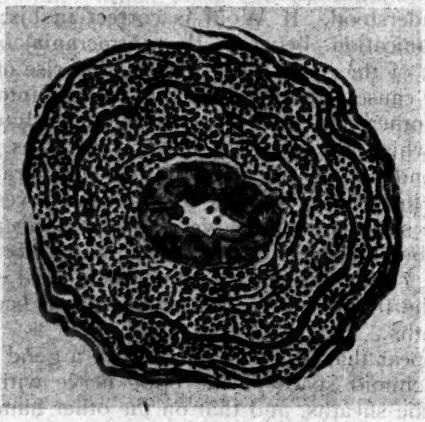
Two points of general resemblance were the presence of ecchymotic discoloration of the optic nerve sheaths, at varying intervals from behind the optic canal to the anterior extremity of the optic nerve, and of an ampulliform distension of the sheaths of the nerves immediately behind the eyeball. Subsequent microscopic examination showed that this distension was usually due to the presence of fluid in the subarachnoid space.

Priestley Smith (1884) was apparently the first to notice the phenomenon of blood in the subdural space associated with fluid distension of the subarachnoid space. That this is not very exceptional is shown by its occurrence in four of my five cases, the fifth case showing complete filling of all the spaces by blood (Compare and contrast Figs. 16 and 22).

The cause and significance of this distension of the sheath are not clearly understood. If Wolff is correct in his finding of a patent communication between the intracranial subarachnoid space and that of the optic nerve, the sudden rise of intracranial pressure could cause cerebro-spinal fluid to pass into and distend the latter. Another consequence would be the early occurrence of papilloedema which has been reported in some cases. The characteristic form and limited extent of the distension cannot be fully explained by its relation to the central retinal vessels as they cross from the sheaths to the nerve. This crossing point is some 12 to 15 mm. behind the globe, while the ampulliform swelling is not more than 7 mm. in length. Some anatomical explanation is required for the failure of the oedema to spread backward along the nerve sheaths.

It would appear that it is possible to have a good deal of blood in the subarachnoid space of the optic nerve without any discoloration of the sheaths, and that on the other hand the familiar blue ecchymotic appearance is due, not so much to the effusion of blood in the subarachnoid space, as to the haemorrhage in or beneath the dural sheath. Even in the intracranial part of the

optic nerve the haemorrhage may be only dural and subdural, and with haemorrhage present in the sheaths both before and behind the optic canal there may be no haemorrhage within the canal itself. This fact of course conflicts with the often repeated statement that the blood in the middle fossa passes, by continuity of the subarachnoid space, through the optic canal and along the course of the orbital portion of the nerve. This view seems to be the result of a too restricted investigation of the pathological material, and Liebrecht seems to be the only observer who has tried to trace the source and distribution of the blood by serial sections. On a superficial view the foregoing statement might seem to be consistent with the appearances in our third case; but examination of serial sections in all the other cases shows that separate effusions of blood occur at varying intervals along the course of the optic nerves, and may be found on the outer surface of the dural sheath, in the dura itself or in the subdural space, in the subarachnoid space and even in the pial sheath. They may occur around the vessels of the circle of Zinn, among the nerve fibres of the optic papilla, between the neuroepithelium and pigmented epithelium of the retina, and in the vitreous; on the episcleral surface of the eyeball, among the orbital fat and muscles, and in the fibrous sheaths surrounding the ciliary ganglion and posterior ciliary nerves. In many of these situations a ruptured vessel has been found in the midst of the haemorrhage, or a vessel with blood effused among the coats of its wall (Figs. 7, 30, 31). The discovery of many such haemorrhages within the

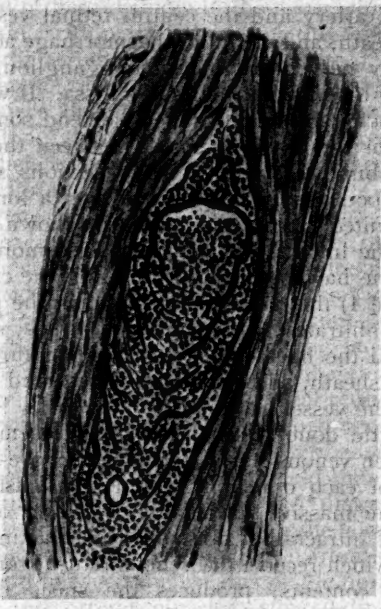


CASE 3.—FIG. 30.

Dissecting haemorrhage in vessel on outside of dural sheath of optic nerve.

chiasma and optic tracts in our second case (Figs. 9 to 14) suggests that if our investigation were to be carried further in similar cases we might find other examples of this distribution.

One would draw attention especially to the sections in Case 5, in which the walls of the central retinal artery and vein were free from haemorrhage in the pial sheath, the perineural spaces and



CASE 5.—FIG. 31.

Dissecting haemorrhage from large vessel in the dural sheath of optic nerve.

the dural sheath; whereas, some distance behind this point, where the vessels emerge from the dural sheath to join the ophthalmic artery and vein, haemorrhage was found in the adventitia of both vessels. Clearly, this could not be explained by pressure on the central vein of the retina where it crosses the perineural spaces, even if there had been evidence of such pressure.

These considerations indicate that we cannot be satisfied with the view that the intracranial haemorrhage passes along the sheaths of the optic nerve, and that it compresses the central retinal vein at its crossing of the perineural spaces and thus leads to haemorrhage into the retina. We must look for a mechanism which will

cover the wide field illustrated by these cases. It is not impossible that distension of the subarachnoid space, either by blood or by serous fluid, could cause a sudden pressure and tension on the central vein as it crosses the space, and thus bring about haemorrhage from any of the tributaries of that vein, but that could only apply to the limited area distal to the crossing of the central vessels.

It is natural that the discovery of haemorrhage surrounding the ophthalmic artery and the central retinal vessels outside and in the dural sheath, and of similar haemorrhage accompanying the posterior ciliary nerves and the ciliary ganglion, should suggest the tracking of blood from the middle fossa. It would be natural, too, for subarachnoid haemorrhage to extend some distance within the space, or for blood to spread between the lamellae of the dural sheath, but the study of serial sections shows that these effusions of blood cannot be followed over a long course. They are of quite limited extent, and cannot be shown to have any connection with the intracranial sheath spaces; moreover, as we saw in regard to the haemorrhage surrounding the ciliary nerves and ganglion (Case 4) the blood is not found in the perineural lymph spaces, but infiltrates the connective tissue sheaths of these structures; and the haemorrhages accompanying the vessels outside the dural sheath, and elsewhere, were found to be splitting up the layers of the vessel wall.

There is little doubt that the multiple haemorrhages are the result of sudden venous stasis, and that they are simultaneous and independent of each other. It would seem justifiable to assume that, when the massive intracranial bleeding occurs, there is a sudden rise of intracranial pressure which, acting upon the veins and sinuses, which receive the venous return from the eyeball and other orbital contents, produces the stasis necessary for the occurrence of the haemorrhages.

The reasons for differences in the distribution of the haemorrhages in different cases must be largely a matter for speculation. Sudden rise of pressure throughout the veins of the eye, the optic nerve and the orbital contents may be supposed to be a common factor, and no doubt the vulnerability of the vessels may be influenced by such constitutional disorders as syphilis, renal disease, or tuberculosis; but for the rest, we must assume the existence of *loci minoris resistentiae* in the walls of the veins. Such *loci* are believed to be prone to occur at the bifurcations and junctions of vessels; and points where the vessels suffer a sudden change of direction, or an alteration of the support afforded by the surrounding tissues, as for example, where the central vessels pass from the optic nerve to its sheaths or from the lamina cribrosa to the retina, may be relatively weak. The condition shown in

Fig. 18 suggests that another such point may lie at the entrance or exit of the central vessels from the dural sheath.

Summary

Five fatal cases of spontaneous subarachnoid haemorrhage are described with special reference to the pathological condition found in the eye and its related structures.

The discovery of haemorrhage in the subarachnoid space at the base of the brain, within the sheaths of the optic nerve, and in many cases in the retina and even in the vitreous, originally led to the belief that the blood passed in a continuous manner in the subarachnoid space through the optic canal into the orbit, and thence either along the course of the central retinal vessels, or by lymph channels accompanying the optic nerve at the periphery of the lamina cribrosa into the retina.

It is now generally agreed that the blood in the cerebral subarachnoid space does not, as a rule, travel through the optic foramen into the subarachnoid space of the optic nerve. It is more generally assumed that haemorrhage occurs in the dural sheath at the apex of the orbit, and tracks forward into the subdural and subarachnoid spaces, and that blood in the latter situation, by putting pressure and tension on the central retinal vein where it crosses the space, causes stasis in the central retinal vein and thus accounts for the retinal and vitreous haemorrhages.

On the basis of the five cases here described it is shown that haemorrhages occur over a much wider field than the optic nerve sheaths, the retina and the vitreous, and that these cannot be accounted for by the assumed pressure on the central retinal vein.

The haemorrhages varied in distribution from case to case, but were found in and between the sheaths of the optic nerve, among the orbital fat and orbital muscles, surrounding the posterior ciliary nerves, ciliary ganglion, ophthalmic and posterior ciliary arteries, and the vessels of the chiasma and optic tracts; as well as beneath the retina, in all its layers, in front of the retina and in the vitreous. Serial sections showed that these were not in continuity, one with another; but were discrete and independent haemorrhages, simultaneous in their occurrence; and it is submitted that the occurrence of such multiple haemorrhages can only be explained by a sudden rise of intracranial pressure causing a stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit.

It is suggested that some of the clinical signs of subarachnoid haemorrhage, such as oculo-motor pareses and disturbances of conjugate movements of the eyes, may be explained by the occurrence, in the midbrain, of haemorrhages similar to those found, in one of our cases, in the chiasma and optic tracts.

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A CASE SHOWING PARTIAL DEFICIENT FUSION OF A MAXILLARY PROCESS WITH LATERAL NASAL PROCESS ON ONE SIDE

BY

G. E. DODDS

WEST AFRICAN MEDICAL SERVICE

BY an odd coincidence I saw the case here described just after I received the January number of the Journal containing an account of cases with malar bone deficiency. As I have access to a very limited literature here I am unable to say much about it.

The patient in this instance is an African baby girl, aged 3 months. The first child of healthy parents who state that there is no history of hereditary defects in the family.

The outstanding features on first viewing the patient were:

1. The distortion of the right nostril with a hiatus showing the interior of the nose—the cleft extending upwards to within about 1 cm. of the inner canthus.

2. The abnormality of the inner canthus—wide obtuse—angled.

With more detailed examination the following additional abnormalities were noted:

(*a*) Upper lids—a small notch—coloboma—at junction of inner with outer two-thirds in each lid—lateral to the puncta.

(*b*) Right lower lid—normal lateral to the punctum while medial to this is a notch the inner side of which is continued in a straight line to the medial end of the upper lid. No caruncle and doubtful plica semilunaris.

(*c*) On the inner side of the left medial canthus is a vertical hole; in appearance like the opening of a sinus. It was a blind shallow pit.

(*d*) The cleft or fissure extends from the lower and lateral margin of the right nasal bone to the point where normally the nostril joins

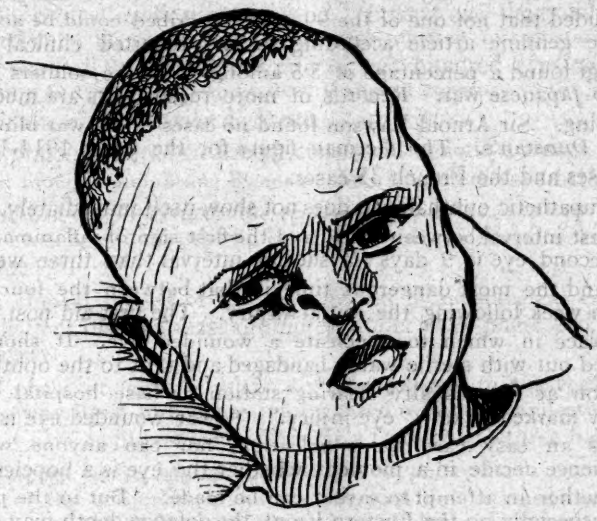

Diagram to show sites of clefts and notches.

the lip. This is merely a cleft in the cartilagenous part of the nose. The surrounding bones appeared normal.

(*e*) Conduction of tears appeared to be normal.

(*f*) Eyes normal in every respect.

(*g*) Ears somewhat large and cup-shaped.

Two points of particular interest arise.

1. What is the significance of the blind pit in the skin on the inner side of the medial canthus? Any embryological explanation?

2. The treatment of this condition. As the completion of the nasal cleft would require a pedicle graft from the arm or forehead it was deemed advisable to wait as it was considered that there would be a better chance of success later on. Is this the correct attitude in cases of this nature?

ANNOTATION

Sympathetic Ophthalmitis in War Injuries

The Czechoslovak Medical Bulletin, No. IV, contains a paper on the subject of war injuries of the eye and sympathetic ophthalmitis by Prof. Loewenstein. Reports of the incidence of sympathetic ophthalmitis in warfare have varied greatly during the past 100 years. In the American Civil War a percentage of 16·1 was recorded and in the Franco-Prussian War of 1870 one of 55·6. In contrast with this figure Dimmer, from the official army report, concluded that not one of the 99 cases described could be accepted as the genuine article according to the reported clinical signs. Masugi found a percentage of 5·5 among Japanese soldiers in the Russo-Japanese war. Records of more recent wars are much less alarming. Sir Arnold Lawson found no cases in his war blind men at St. Dunstan's. The German figure for the years 1914-18 was 13 cases and the French 35 cases.

Sympathetic ophthalmitis does not show itself immediately. The shortest interval between injury and the first sign of inflammation in the second eye is 9 days; a shorter interval than three weeks is rare and the most dangerous time is that between the fourth and eighth week following the initial injury. The first-aid post is not the place in which to enucleate a wounded eye. It should be washed out with sterile water, bandaged and sent to the ophthalmic surgeon at the casualty clearing station or base hospital with a clearly marked ticket, "eye injury." A war wounded eye is by no means an easy case for enucleation, nor can anyone without experience decide in a moment whether the eye is a hopeless case or whether an attempt to save it can be made. But in the present war, especially on the Eastern Front, the defence depth may extend to thirty or forty miles on either side of the front line. "A time evacuation factor" has to be considered and if this appears likely to exceed 3-4 days a suspected eye should be excised.

A case bearing on early excision in warfare sticks in the writer's memory. It was that of an N.C.O. who received a contusion at the first battle of Ypres. When the writer saw him at a civilian hospital in England his eye was quite white and there was some vision, about 6/36 according to memory. The patient said that he had been advised to have his eye removed but, as he could see with it, he had refused. The condition found was one of subluxation of the lens and with an appropriate cylinder vision of 6/6 was obtained.

ABSTRACTS

I.—MISCELLANEOUS

(1) **Bonnefon (Bordeaux). — L'Oeil Ypérite. Son Traitement.** *Gazette hebd. des sc. med. de Bordeaux.* Nos. 11 and 12, Mars. 1939.

(1) In September, 1918, at a French military hospital in France **Bonnefon**, the only oculist, without medical assistance and without specially trained medical orderlies, had to treat one thousand eight hundred men in whom both eyes were affected by mustard gas. These were all treated at the rate of two hundred eyes an hour according to the method to be described and contrary to the official instructions of the Service de Santé.

The principle of the treatment consists in osmotic drainage of the affected mucous membrane by means of a hypertonic eye-bath.

In view of its importance the reprint of the author's article in the Gazette has been deposited in the Library of the Royal Society of Medicine.

The clinical picture of the eye affected by mustard gas exhibits three distinct phases, phase of impregnation, acute phase and phase of recovery.

The phase of onset is silent, the eye exposed to the vesicating gas allowing itself to become impregnated without manifesting the slightest pain or discomfort. This lasts from one to three hours.

The acute phase begins suddenly by hypersecretion of all the glands of the conjunctiva and the lacrimal flow washes away the secretions of the Meibomian glands. Stabbing pain accompanied by photophobia begins and in a few minutes the clinical picture is completed by such oedema of the eyelids as to close the palpebral fissure. The eye is now almost exactly like that of an infant with gonococcal ophthalmia neonatorum, except that there is no pus. When it is possible to open the palpebral fissure a yellowish blistering fluid spurts out. This phase lasts from two to five days but may be shortened by judicious treatment.

The phase of recovery is characterized by a progressive diminution of the palpebral oedema, persistence of the lacrimal secretion and photophobia with marked dilatation of the submucous capillary network. This phase may last for weeks or months. Sometimes secondary infections of the conjunctiva or cornea may occur but these have no direct relation with the vesicating action of the gas

which has long disappeared. This phase of sequelae may be shortened or prevented by further skilled therapy.

The mildness of the ocular lesions produced by blistering gases as compared with the destructive effect on other parts of the body is indisputable.

In contact with the tissues mustard gas or dichlorethyl sulphide undergoes hydration with formation of hydrochloric acid. Severe pain is experienced and the defensive mechanism of the body is brought into play. No organ is more exposed or better protected than the eye; there is the reflex flow of tears which clears away the caustic from the conjunctival fornices and from the ducts of glands; osmotic action which fills up the subcutaneous lymph spaces; dialysis of fluid from the submucous capillaries. This superficial and deep lavage prevents any eruption of blisters.

In 1917 the author saw in the narrow valley of Belrupt, near Verdun, a battery of horse artillery which had been shelled with mustard gas the day before. A number of the horses exhibited absolutely opaque corneae as result of the gas while none of the gunners had to be sent to hospital. He observes that the horse and the rabbit have no lacrimal secretion nor peri-ocular lymphatic defences, which in man are present and render the eyes less liable to damage by gas.

It is therefore reasonable to conclude that since the natural defences of the ocular adnexa are sufficient to protect the eyes in slight cases of damage by gas, in severe cases the aim should be to reinforce the natural mechanism.

The author carried this out after another heavier gas attack as soon as the casualties arrived at the First Aid Post by cleaning up the eyelashes and applying by means of an eye-bath a warm hypertonic solution. This consisted of:—

Saturated solution of sodium sulphate 800 grammes (approx. 1½ pints); Syrupus simplex 200 grammes (approx. 1/3 pint).

However great may be the palpebral oedema this solution enters the palpebral fissure and penetrates to the fornices and osmotic drainage by the lymphatic exudation occurs. After ten minutes of immersion the fluid in the eyebath becomes acid proving the efficacy of the treatment. With two hundred eye-baths and four First Aid Assistants fifteen hundred eyes were bathed twice a day.

This osmotic treatment should be continued not only during the acute stage but also as long as there is any photophobia and lacrimation.

When an analgesic is required the author recommends the instillation of a 2 per cent. solution of dionine.

The treatment for gassed eyes given in the official "Medical Manual of Chemical Warfare" 1943, pp. 16 and 19, envisages the flushing of the conjunctival sac. with water if this can be done *within* five minutes, the instillation of atropine as a mydriatic, and then of albucid soluble 2·5 per cent. as an antiseptic. If the discharge is excessive the sac may be washed out with normal saline solution. Cocaine as an analgesic must never be used as it will cause damage to the corneal epithelium. Liquid paraffin and oily drops must never be used while there is any possibility of any dichlorethyl sulphide remaining in the sac, as they are solvents thereof. The reviewer's criticism of this treatment is that if atropine is used as a routine for slight casualties the recipient will be put out of action for fighting for a week or a fortnight, while in a case of only moderate severity it is impossible to introduce irrigation fluid into the conjunctival sac on account of the tight closure of the lids.

A. F. MACCALLAN.

(2) **Métivier, V. M. (Trinidad).—Eye disease due to vitamin deficiency in Trinidad.** *Amer. Jl. Ophthal.*, November, 1941.

(2) **Métivier,** in an able paper, discusses such conditions as tropical nutritional amblyopia, essential corneal epithelial dystrophy and conjunctival bleeding in the new born. He says that vitamin A deficiency is of little importance in Trinidad, but that "the ocular manifestations of vitamin B deficiency, B_2-complex syndromes in particular, provide a good deal to think about and do." From his experience he finds that such conditions as photophobia, hazy vision, lacrimation, failure of central visual acuity and partial optic atrophy appear to fit into two distinct clinical pictures that are the result of failure of vitamin B_2 to maintain the due nutrition of the cornea and optic nerve respectively. Table I gives vitamin-deficiency statistics between the dates 1888 and 1940. Table II gives the known vitamin-deficiency factors involved in the production of different conditions.

Twelve illustrative case records with commentaries are given and essential corneal epithelial dystrophy is described and illustrated with a couple of good plates. A short section is devoted to cases treated with lactoflavine (riboflavine) and another on conjunctival bleeding in the new born, of which subject the author gave the first account in a letter to the *Lancet* in 1937. Summarising he says that the "title Tropical nutritional amblyopia is suggested for the disease which results from failure of vitamin B_2 to maintain the due nutrition of the optic nerve, either directly or through retinal elements when the nutrition of the cornea suffers from deficiency of vitamin B_2, ariboflavinosis in particular, essential corneal epithelial

dystrophy is the disease which develops in Trinidad" . . . special attention is called to a dry parched-skin condition which is seen in patients suffering from eye disease due to vitamin-B_2 deficiency.

A good bibliography is appended.

The delay in publishing this abstract is explained by the fact that the paper was mislaid for which apologies are tendered.

R. R. J.

(3) **Scheffler, Milton M. (Chicago).—Visual loss following distant haemorrhage.** *Arch. of Ophthal.*, Vol. XXIX (old series, Vol. LXXXVI), p. 449, No. 3, March, 1943.

(3) **Scheffler** divides the occurrence of visual loss following distant haemorrhage into several categories. The first is functional, generally following closely or immediately after the haemorrhage, and is probably due to anaemia of the cortical centres. Recovery for the most part begins immediately and is usually complete. The second variety is rare—none in 60,000 cases of haemorrhage (Haab), one in 30,000 cases (Dufour). It was first referred to by Hippocrates and occurs most commonly in those who have previously had recurrent haemorrhages from gastric and duodenal ulcer, pulmonary tuberculosis, uterine disturbances, etc. The resulting blindness is bilateral in 85 per cent. of cases. No single factor is responsible for the visual loss in all cases, but the most important is anaemia resulting from loss of blood and causing anoxaemia of the tissues. When recovery ensues, there have been no complications, but when it does not, it is assumed that the anaemia allows toxins which were present, to poison the ganglion cells of the retina and bring about degeneration of the nerve fibres. Holden's experimental work supports this. He found that one to two days after bleeding dogs and rabbits, oedema was present in the ganglion cells and nerve fibres of the retina, which in two weeks went on to advanced degenerative changes. Goerlitz confirmed these findings in post-mortem examinations of human beings. Repeated transfusions of blood are sometimes of value in restoring vision, but if a toxic factor is present as well as the anaemia, the degenerative changes are irreversible and no recovery occurs. Ophthalmoscopically, there is, in the early stages, papilloedema of varying degree followed later by pallor of the discs and alteration of the vessels. In severe cases, there may be some pigmentary degeneration of the retina. The field changes are variable, but the commonest finding is peripheral contraction. Clinically, blurred vision occurs at the time of, or immediately after the haemorrhages in 20 per cent. of cases, within two days in 33 per cent., in 40 per cent. within 3-16 days, and in 7 per cent. after sixteen days. Some improvement occurs in 50 per cent. of cases, but normal vision is regained in only 10-12 per cent.

Apart from retinal damage by anaemia and toxins, vision may be affected (i) by haemorrhage into the sheath or interstitial tissue of the optic nerve, or (ii) by embolism or thrombosis of the retinal vessels. The article concludes with the report of a case of unilateral visual loss with bilateral field impairment in a case of haematemesis from peptic ulcer.

F. A. W-N.

(4) **Kolen, A. A.—Soviet military ophthalmology.** *Lancet*, June 26, 1943.

(4) **Kolen,** who is head of the Novosibersk Eye Clinic, contributes an interesting account of Russian war ophthalmology in the second world war. He makes the point that it is a good thing to group the departments of ophthalmology, otolaryngology and stomatology together in base hospitals. In modern warfare injuries are nearly always multiple and wounds of the eye, nose, accessory sinuses and mouth are often met with in the same subject and need the attention of specialists in all three branches.

Much of the paper deals with plastic surgery and there is a section on the importance of the ciliary body and another on transfusion. In the Novosibersk eye clinic they have had good results in the extraction of intra-ocular foreign bodies coupled with excision of prolapse of the ciliary body and many eyes have thus been saved which in earlier years would have been excised at once.

It is most satisfactory to know that "the high efficiency of the ophthalmic service has assured the return of a high percentage of ocular casualties to the ranks of the Red Army and to the labour front." The article is one which should be widely read by ophthalmologists everywhere.

R. R. J.

(5) **Berens, C. (New York).—Plastic suture plates for the closure of wounds in certain ophthalmic operations.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 981, 1942.

(5) **Berens** uses quadrangular and circular plates made of lucite or plexiglass, 5 mm. by 2 mm. by 3 mm., with two holes drilled for the passage of atraumatic needles. The edges are slightly rounded. They do not irritate the tissues, nor do they absorb serum or pus. He employs these for closing small wounds of the cornea and conjunctiva and for muscle and plastic operations.

H. B. STALLARD.

(6) **Carter, L. F. (Detroit).—A flexible tip for the giant magnet.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 986, 1942.

(6) **Carter** describes the advantages of using a flexible tip for the giant magnet. The instrument is fitted with either a screw-in

or a plug-in attachment to replace the regulation tip in the core of the magnet. From this extends a five inch flexible steel coil spring to the free end of which may be attached any one of 3 types of screw-on tips of varying degrees of magnetic pull.

H. B. STALLARD.

(7) **Mackenzie, C. M. (Spokane, Washington).—Fascial suspension of the eye.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 977, 1942.

(7) **Mackenzie** comments on the deformity of downward and outward displacement of the eye after excision of the maxilla for a malignant neoplasm. He has used in two such cases a strip of fascia lata, taken from the upper part of the thigh, and suspended like a hammock between the lateral and medial canthal ligaments, to which it is secured by mattress sutures. The fascial string contracts subsequently and maintains the eye satisfactory in position.

H. B. STALLARD.

II.—PATHOLOGY

(1) **Ash, J. E. and Wilder, H. C. (Washington, D.C.).—Epithelial tumour of the limbus.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 926, 1942.

(1) **Ash** and **Wilder** have studied 93 epithelial tumours arising at or near the limbus. Of these 83 occurred in white persons and 10 in negroes, 78 were males and 15 females. The highest incidence occurred between the ages of 50 and 80 years. In 8 instances, 4 papillomas and 4 carcinomas, the neoplasms appeared at the site of and immediately following injury. Metaplasia of the corneal epithelium was shown histologically in 31 out 93 cases, and in 18 additional cases there was clinical evidence of corneal involvement.

Epidermatization, an important precursor of tumour development, was seen in 12 of the 28 papillomas and 35 of the 48 carcinomas. Of the latter 39 were of the squamous-celled type. Extension through the sclera into the ciliary body and iris and metastases are rare. On this account local excision of the neoplasm is justifiable. The authors consider that irradiation before or after excision of the growth is unnecessary. Enucleation of the eye appears to be justified only where there is already appreciable involvement of the cornea or after local excision has failed.

H. B. STALLARD.

(2) **Callender, G. R., Wilder, H. C. and Ash, J. E. (Washington, D.C.).—Five hundred melanomas of the choroid and ciliary body followed five years or longer.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 962, 1942.

(2) **Callender, Wilder** and **Ash** give some interesting statistical facts about the progress of malignant melanoma of the choroid and ciliary body. In their series of 500 cases followed over 5 years the incidence of males is slightly higher than females and so is the death rate among the former sex. The youngest patient was 17 years and the oldest 88. The highest incidence of the disease was in the sixth decade of life and the highest death rate in the eighth and ninth decades. The worst prognosis is in the epithelioid cell type and in tumours possessing no argyrophil fibres. The paper contains tables showing the outcome according to cell type, argyrophil fibre content and pigment content. The authors comment that there is not enough variation in death percentages to conclude that heavy pigmentation makes the prognosis more serious.

In the 500 cases there were 239 deaths from metastases, that is 48 per cent. This percentage increased to 66 in 200 of these cases followed for 10 years or longer.

H. B. STALLARD.

(3) **Wetzel, J. O. (Lansing, Michigan).—Tetanus following eye injury.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 933, 1942.

(3) **Wetzel** comments on the rarity of tetanus as a complication of an eye injury. 30 cases had been reported in the literature up to 1941. The author's case was a farmer injured by a nail striking his eye whilst repairing a barn. He received 1,500 units of tetanus antitoxin. 12 days after the injury he developed tetanus. 375,000 units of antitoxin were administered by muscular, spinal and intravenous injections. The injured eye was removed on the 26th day after the accident and he recovered.

All cases reported in the literature were associated either with horses or contaminated earth. It is interesting that none were war wounds. The average time of onset was 12 days after the accident. The clinical signs consist of trismus, facial paralysis on the side of the injured eye, sometimes ocular palsies on one or both sides, the third and the sixth cranial nerves being more frequently involved. 80 per cent. of cases are fatal. The author's patient is the only one, reported in the literature, who received prophylactic tetanus antitoxin and recovered, so he advocates it.

H. B. STALLARD.

(4) **Brinckerhoff, Lt. A. J. (San Francisco).—Actinomycosis of the inferior lacrimal canaliculus.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 978, 1942.

(4) **Brinckerhoff** describes a case of actinomycosis in a 19 year old boy which appeared as a swelling in the tarsus, medial to the lower punctum and communicating with a patent lower canaliculus. The swelling contained yellowish-green granules. 0·5 per cent. sulphanilamide ointment was used thrice daily and was thought to expedite healing.

H. B. STALLARD.

(5) **Espildora, C. and Coutts, W. E. (Santiago, Chile).—Lymphogranuloma venereum lesions of the eyes.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 916, 1942.

(5) **Espildora** and **Coutts** state that lymphogranuloma-venereum is probably analogous to syphilis. Their paper consists mainly of extracts from 49 references in the literature about this disease. It seems from these writings that any part of the eye may be affected during the course of this disease. This paper contains some irrelevant matter and the substance of some of the assumptions is unconvincing.

H. B. STALLARD.

(6) **Lyon, N. B. (South Bend, Indiana).—Case of conjunctivitis caused by staphylococcus aureus giving a strongly positive coagulase reaction.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 983, 1942.

(6) **Lyon** describes a case in which a pure growth of staphylococcus aureus cultured on blood agar, after swabbing a patient with acute conjunctivitis, was used in the coagulase test of Chapman, Berens, Peters and Curcio. A loop-ful of the culture growth was emulsified in 0·5 cc. of citrated normal human plasma and placed in an incubator. One hour later the plasma formed a firm clot. The conjunctivae recovered quickly after using 5 per cent. sulphathiazole drops 2 hourly by day and 5 per cent. sulphathiazole ointment night and morning.

H. B. STALLARD.

III.—MUSCLES

(1) **Hughes, Wendell L. (Hempstead, Long Island) and Bogart, D. W. (New York).—Recession of the trochlea in overaction of the superior oblique.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 911, 1942.

(1) **Hughes** and **Bogart's** operation of recession of the trochlea in overaction of the superior oblique consists in incising the periosteum of the orbital roof for 1 cm. concentric with the supra-orbital margin and thus immediately above the pulley of the muscle. The periosteum is then incised antero-posteriorly for 1·5 cm. from the medial and lateral extremities of the 1 cm. incision described above. The periosteum with the pulley of the superior oblique is then stripped from the orbital roof and pushed posteriorly. Two cases are described. The hypotropia and intorsion of the affected eye was reduced and the patients relieved of their symptoms.

H. B. STALLARD.

(2) **Leinfelder, P. J. and Black, Nelson M. (Iowa City).—Experimental transposition of extra-ocular muscles in monkeys.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 974, 1942.

(2) **Leinfelder** and **Black** describe the results of a series of experiments on monkeys in which the tendon of the superior oblique muscle was either tenotomized or resected either before or after transposition operations had been made on the recti muscles of one or both eyes. Recovery of co-ordination occurred in the majority. They consider that maintenance and perhaps recovery of co-ordinated rotations depends upon vision and propioception, but vision alone is not the factor in recovery for this occurred in some animals kept in the dark after the transposition operations.

Tenotomy of the superior oblique was tolerated without loss of co-ordination. This muscle does not appear to have any special co-ordinating function that cannot be ascribed to any other muscle.

H. B. STALLARD.

IV.—MEDICAL OPHTHALMOLOGY

(1) **Fagin, D. (Detroit).—Chronic ophthalmoplegia externa.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 968, 1942.

(1) **Fagin** reports a case of chronic opthalmoplegia externa occurring in a male aged 45 years, who had had ptosis of the left

upper lid for 30 years and of the right for 6 years and also restricted movements of both eyes. He became uncommunicative, lethargic, disorientated in time and place, and had fleeting delusions about being followed and watched. The author presumes that progressive nuclear degeneration was the underlying pathological process in this case. A transient psychosis accounted for the mental symptoms from which he recovered after rest.

The author gives a classification of the causes of chronic external ophthalmoplegia.

H. B. STALLARD.

(2) **Fox, S. A. and Gelfand, N. L. (New York).—Bilateral exudative retinitis in a case of cerebral haemorrhage: chrysotherapy with recovery.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 984, 1942.

(2) **Fox** and **Gelfand** describe the case of a white male, aged 40 years, with bilateral exudative retinitis and cerebral haemorrhage of sudden onset. Six months after this catastrophe there had been no visual improvement and it seemed that blindness threatened the patient. 23 weekly intravenous injections of gold sodium thiosulphate were given. The first was 10 mg. and the next 6 injections 25 mg. each, and the remainder 50 mg. each. There was no untoward reaction during this treatment. Vision improved to R.V. counting fingers at 3 feet and L.V. 20/400 after the sixth injection and was ultimately 20/20 in both eyes. The authors comment that visual improvement may have been coincidental.

H. B. STALLARD.

V.—GLAUCOMA

Barkan, O. (San Francisco).—Operation for congenital glaucoma. *Amer. Jl. Ophthal.*, Vol. XXV, p. 552, 1942.

Barkan describes a series of 17 goniotomy operations in which the intra-ocular pressure was made normal in 16 and visual function maintained in 14. He stresses the importance of early operation before Schlemm's canal has been occluded by the effects of sustained raised intra-ocular pressure. He quotes other observers who found Schlemm's canal open in 75 per cent. of early cases. In the operation of goniotomy it is not necessary to remove the obstructive tissue in the filtration angle for more than one fourth to one third of the circumference. In the author's series the average age at the time of operation was three months.

When permanent obliteration of the canal has occurred iridencleisis is indicated. The latter is also done in cases where goniotomy has proved ineffective.

H. B. STALLARD.

VI.—TRACHOMA

(1) **Poleff, L. (Morocco).—État actuel du problème d'étiologie du trachome.** *Arch. f. d. ges. Virusforschung.*, Band II, Heft 3, Wien, Springer-Verlag, 1942.

(1) **Poleff** gives an outline of the researches as to the aetiology of trachoma carried out by all known workers. There is a copious bibliography which is useful. The essay is in French and a copy has been deposited in the Library of the Royal Society of Medicine. A few excerps may be quoted.

The virus inclusion bodies first described by Halberstaedter and Prowaczek can be found in all active stages of the disease and also in many patients in whom the disease is entirely quiescent, and in whom clinical symptoms are entirely absent. From them baboons can be inoculated with production of a conjunctivitis, differing from human trachoma in that neovascularization of the normally clear cornea or pannus never occurs, and in that the virus inclusion bodies cannot be found in the epithelial cells of the baboon's conjunctiva. However, if the conjunctiva of a man is inoculated from the baboon a typical trachoma develops with presence of the virus inclusion bodies.

By some authors the trachoma virus is classified among the Rickettsiae on the strength of the Weil-Felix reaction being present (that is agglutination of Proteus OX). However, the reviewer must remark that this is entirely denied by van Rooyen and Rhodes (Virus and Diseases of Man, Oxford University Press, 1940). One of the criteria of Rickettsial disease is that there must be an arthropod host. Such is not the case in trachoma, *pace* Cuenod and Nataf.

A. F. MACCALLAN.

(2) **Poleff, L. (Morocco).—Sulfamides dans le traitment du trachome.** La Presse Medicale, Paris, 28 Fevrier-2 Mars, 1940.

(2) **Poleff** is well known for research work on the aetiological agent of trachoma, which he has carried on for some years at the Institute of Hygiene at Rabat, Morocco. The present paper is an experimental and clinical study of the treatment of trachoma by drugs of the sulphonamide group. Among these were M & B 693, prontosil, soludagenan, ulirone, rhodilone and septoplix.

He found that the conjunctival flora were uninfluenced either by instillation or by subconjunctival injection confirming the results obtained by Wolff and Julius (*Ann. de l'Inst. Pasteur*, 1939, Vol. LXII, p. 616), which showed that sulphanilamide is not an ordinary antiseptic.

Virus inclusion bodies or Halberstaedter-Prowaczek Körperchen which are found in the conjunctival epithelial cells in trachoma and are believed to be the exciting agent of the disease were uninfluenced by the exhibition of the drugs.

However, when the author used intramuscular injections he found that the bacterial flora of the conjunctiva diminished and, when virus inclusion bodies were not too numerous to start with that they disappeared. When this form of treatment is added to the usual local applications of copper sulphate, etc., his therapeutic results were better than when using local treatment alone.

He gives a warning that the local use of sulphonamides may result in a dilatation of the circumcorneal blood vessels and cannot be recommended.

A. F. MACCALLAN.

BOOK NOTICES

Transactions of the Ophthalmological Society of the United Kingdom. Vol. LXII. London: J. and A. Churchill. 1943.

The sixty-second volume of the *Transactions* is a rather slim volume similar to those which we have become accustomed to in war time. It contains the discussion and papers read at the Annual Congress of the Society last year, together with the papers of the Oxford Congress and those of the North of England and Irish Ophthalmological Societies. The volume itself compares very favourably with its predecessors and contains much of interest. The chief change in it is due to the setting of the discussions after the various papers in 9-point type. This has effected a considerable saving in paper and we found the result quite legible. It was a pity that the publication could not have been effected earlier, but this was not possible in existing circumstances.

Internal Diseases of the Eye and Atlas of Ophthalmology. By By M. URIBE TRONCOSO, M.D. Pp. 530, with 240 engravings, including 95 figures; 82 full-page colour plates. Philadelphia: F. A. Davis Company. 1942. Price $15.

The first edition of this excellent book appeared in 1937 and was reviewed in our columns. It was twice reprinted in the year of

issue and again in 1938 before the present edition was published. This edition contains about 20 more pages and two more illustrations than the first. The author adopts the scientific method of emphasizing the pathological changes and physio-pathology of the disease in endeavouring to deduce the symptoms from the lesions. He also contributes an admirable chapter on the importance of perimetry and has introduced such "new names as retinosis, choroidosis and perivascular pigment." The reproductions of X-rays are very good indeed, particularly one which shows a shadow of calcium deposits in a case of glioma retinae. In the treatment of this condition attention is drawn to the work of Foster Moore, Stallard and Milner in connexion with the implantation of radon seeds. The colour plates are excellent. The book has obviously found favour in the United States. We hope it will become equally well known in this country as we consider it one of the very best books of its kind on the market. Messrs. H. K. Lewis & Co. are the English agents for supplying it.

OBITUARY

LESLIE BUCHANAN

THE recent death of Dr. Leslie Buchanan leaves a gap in the ranks of British ophthalmologists, and especially to his Glasgow colleagues will bring a sense of personal loss,

Born in 1868, he received his medical training in Glasgow University, where he graduated M.B., C.M., in 1898. As House Surgeon in the Glasgow Eye Infirmary, from 1891 to 1892, he decided to devote himself to ophthalmology, and having joined the visiting staff of the Infirmary, he was, in succession, Assistant Surgeon and Surgeon, retiring from the latter post soon after the Great War. For some years he held the post of pathologist to the Eye Infirmary, and in histology and photomicrography he found his chief recreation, inspired, no doubt, by the enthusiasm of his former chief, Dr. Thomas Reid.

He became a member of the Ophthalmological Society in 1901, and during the succeeding fifteen or sixteen years, made many contributions to the Transactions of the Society, mainly on pathological topics. Of outstanding interest and value were his papers on the histology of uveal disease and on birth injuries of the eye. His work on birth injury of the cornea, in conjunction with Ernest Thomson, established our knowledge of the pathology of this condition.

For many years Leslie Buchanan played an important part in the teaching of ophthalmology, and many oculists practising in Glasgow and elsewhere, owe their early training, both pathological and clinical, to his encouragement and example. His assistants found in him an ideal chief—kindly and considerate, with a fine natural dignity and courtesy. He was modest and without affectation, and utter sincerity was the keynote of his character. To his colleagues and to his many friends, his name will be a fragrant memory.

NOTES

University of Glasgow Department of Ophthalmology Autumn Term 1943

IN September a series of meetings will be held in the Eye Department, Western Infirmary, on Wednesdays at 8 p.m. The general arrangements will be similar to the series held last year. Tea will be served after the paper and a discussion will follow. The meetings will be open to all medical practitioners and senior students interested in Ophthalmology.

September 8, Dr. J. B. Gaylor—"The fifth nerve and the cornea"; September 15, Professor Loewenstein—"Herpes of the cornea"; September 22, Dr. J. M. Robson—"Experimental corneal ulcers"; September 29, Professor W. J. B. Riddell—"Chemical injuries of the cornea."

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The Royal Eye Hospital: Clinical Society

A meeting of the Royal Eye Hospital Clinical Society will be held at the Hospital on Friday, September 24, at 5.30 p.m. when a talk will be given by Mr. V. E. Negus, M.S., F.R.C.S., on the following subject:—"The Relationships of Ophthalmology and Rhinology."

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Special Notice

WE are asked by the Ministry of Information to state that the fact that goods made of raw materials in short supply owing to war conditions are advertised in this journal should not be taken as an indication that they are necessarily available for export.